Abrupt Flow Arrest in the Internal Carotid Artery during Carotid Artery Stenting Using the Parodi Anti-Emboli System

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Key words: carotid stenting, cerebral protection, Parodi anti-emboli system

Summary

We report a rare complication of carotid artery stenting (CAS) in a patient with severe carotid artery stenosis. CAS with a protection of the Parodi Anti-Emboli System was complicated by an abrupt flow arrest in the internal carotid artery before the guidewire passage through the stenotic site.

Introduction

Cerebral protection devices during carotid artery stenting (CAS) significantly decrease the incidence of periprocedural complications [1-3]. Currently, three temporary cerebral protection systems are available: balloon occlusion, filtration baskets and flow reversal of the carotid artery. In particular, the temporary flow reversal of the carotid artery during the procedure using the Parodi Anti-Emboli System (PAES: ArteriA, SanFrancisco, CA, USA) seems a viable solution to prevent the possibility of a distal embolism before the passage of the guidewire through the site of stenosis [4]. We report a rare complication of CAS with the protection of PAES.

Case Report

A 67-year-old, right-handed man had a cerebral infarction five years ago. He was referred to us for a diagnostic angiography and endovascular treatment if applicable. Neurological examination revealed right hemiparesis and left visual acuity loss due to ischemic oculopathy. He had been taking aspirin 200 mg and ticlopidine 200 mg daily after a stroke. Cormobidities included hypertension, hyperlipidemia, and diabetes mellitus. He underwent a coronary artery bypass graft 13 years ago. Diagnostic angiography confirmed severe stenosis of the origin of the left internal carotid artery (ICA), NASCET 99% stenosis (NASCET5). Contrast medium ascended very slowly in the collapsed cervical ICA (figure 1).

The intracranial arteries were mainly supplied by the ophthalmic artery as a collateral pathway (figure 2). Cerebral blood flow study by 99 mTc-ECD with diamox administration demonstrated poor vascular reserve capacity of the left ICA territory. After written informed consent, CAS was performed under local anesthesia. PAES was used for the purpose of cerebral protection. After arterial access was obtained, 5,000 units of heparin were injected followed by an infusion of 100 units, adjusted to maintain an activated clotting time of more than 300 seconds. A 10.5 F LILAC balloon mounted guiding catheter and CATTLEYA balloon mounted catheter were advanced into the left common carotid artery (CCA) and the left external carotid artery (ECA), respectively. After the inflation of a balloon placed in the CCA and ECA, flow reversal into the guiding

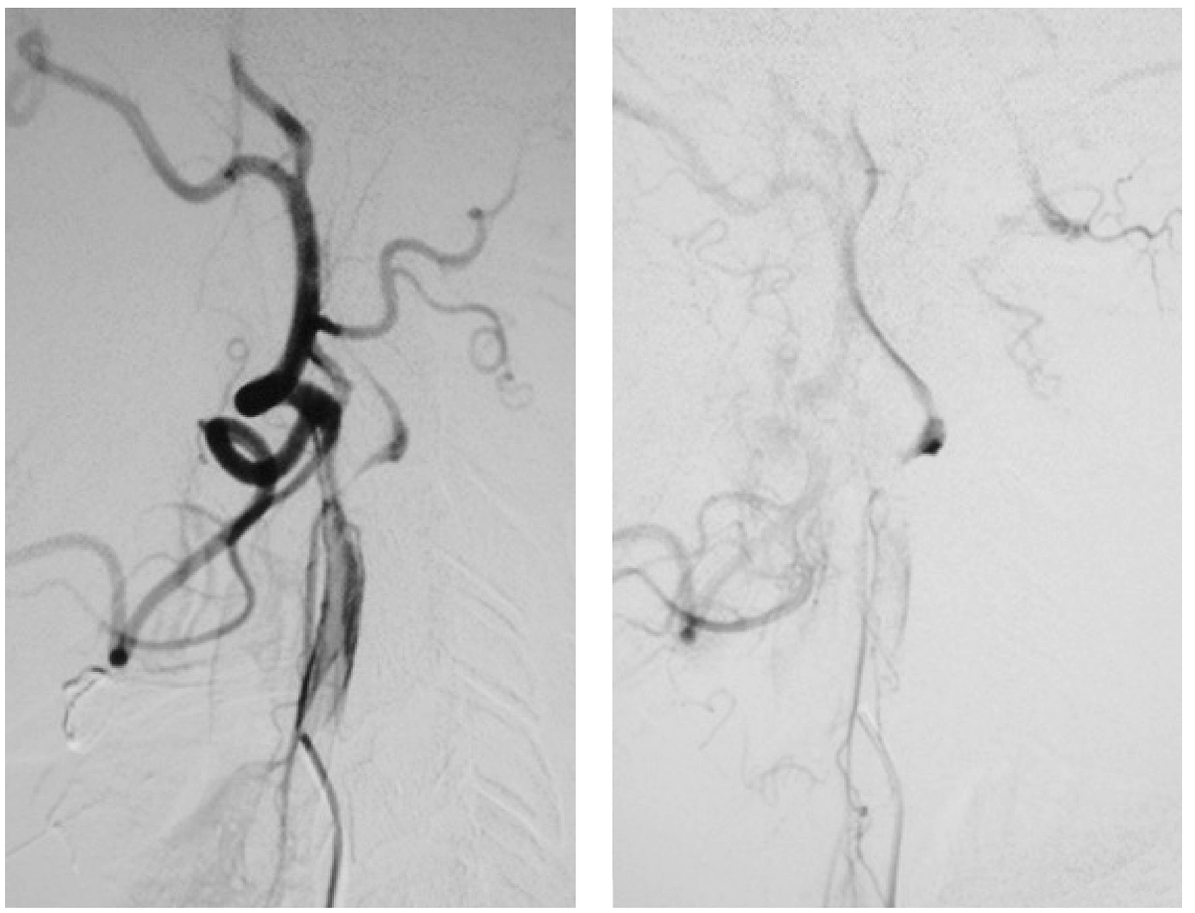


Figure 1 Preprocedural left common carotid angiogram showing a severe stenosis of the internal carotid artery. A) Early phase, B) Late phase.

catheter was recognized under fluoroscopy (figure 3). Although the passage of the site of stenosis, by either 0.018-inch SV wire (Cordis Neurovascular, Miami, FL) or 0.014-inch Essence wire (Cordis Neurovascular, Miami, FL) was attempted using digital road-mapping, we could not cross the lesion. Ten minutes later, the patient presented consciousness disturbance, right hemiplegia and aphasia. We thought that a neurological deficit had occurred due to the flow reversal. Although the balloons placed in the common carotid artery and external carotid artery were deflated, neurological deficit was not improved. Left common carotid artery injection demonstrated the arrest of antegrade flow in the left internal carotid artery with pooling of contrast medium (figure 4).

A microcatheter (Transit 18; Cordis Neurovascular, Miami, FL) and a 0.012-inch GT wire (Terumo, Japan) assembly system were used to cross the stenosis. Fortunately, a 0.012-inch GT wire was able to cross the lesion aided by the microcatheter. Subsequently the microcatheter was advanced coaxially to the distal portion of the left internal carotid artery and a 0.018-inch SV wire was advanced to the distal ICA through the microcatheter. After predilation using a Savvy 3.5 x 20 mm balloon catheter (Cordis Neurovascular, Miami, FL), a self-expanding stent, 7 x 40 mm SMARTeR (Cordis Neurovascular, Miami, FL), was deployed under no cerebral protection. Postdilation was not performed. Poststenting carotid and cerebral angiography demonstrated antegrade blood flow in the left ICA and disappearance of extracranial-intracranial collateral pathway (figure 5). Systemic heparinization of 500 units/h continued for 48 hours. The patient's neurological deficits disappeared completely two hours

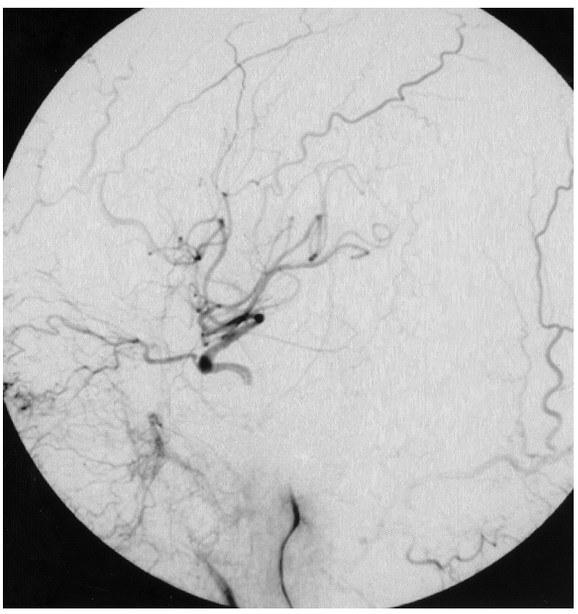
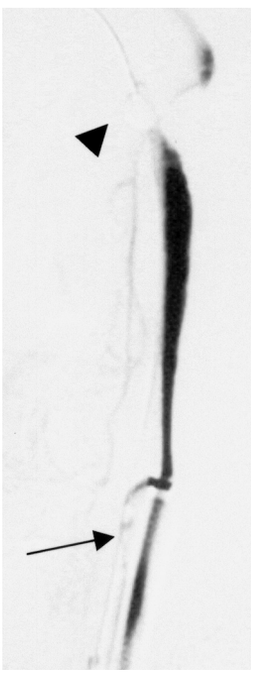


Figure 2 Intracranial arteries were mainly supplied by the ophthalmic artery as a collateral pathway.

Figure 3 Retrograde flow in the ICA during PAES deployment. Arrowhead points to the ECA balloon, arrow to the balloon positioned around the guiding catheter. Non-contrasted blood is pushing the contrast agent out into the guiding catheter.



after the intervention. Diffusion-weighted magnetic resonance imaging was performed on the third day post-treatment, revealing few asymptomatic ischemic lesions. Even though severe headache and ocular pain was recognized during the two days post-treatment, suggesting a hyperperfusion syndrome, the patient was discharged without newly developed neurological sequelae and a daily maintenance dose of 200 mg aspirin and 100 mg ticlopidine.

Discussion

The outcome of CAS may be dramatically changed by the use of cerebral protection devices, as suggested by several recent clinical studies [1-3,6-8]. Although distal protection devices such as balloon occlusion catheters or filters have been widely used, these devices must cross the lesion, which can account for about 12% of all emboli generated during a procedure [9]. The PAES produces retrograde carotid artery flow before the lesion is crossed, providing an extra degree of safety not available with existing balloon occlusion catheters or filters.4 As the degree of ICA stenosis was very severe in our case, the passage of the lesion without protection carried a high risk of distal embolism. Therefore, we preferred to use the PAES. Although the cause of flow arrest in the

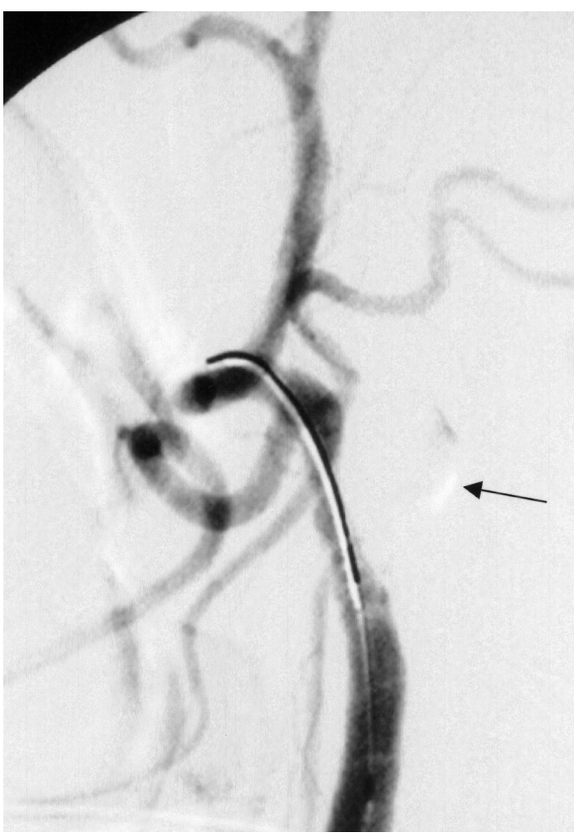


Figure 4 After deflation of occlusion balloons, the carotid injection shows the arrest of antegrade flow in the ICA. Note the subtracted contrast medium in the ICA.

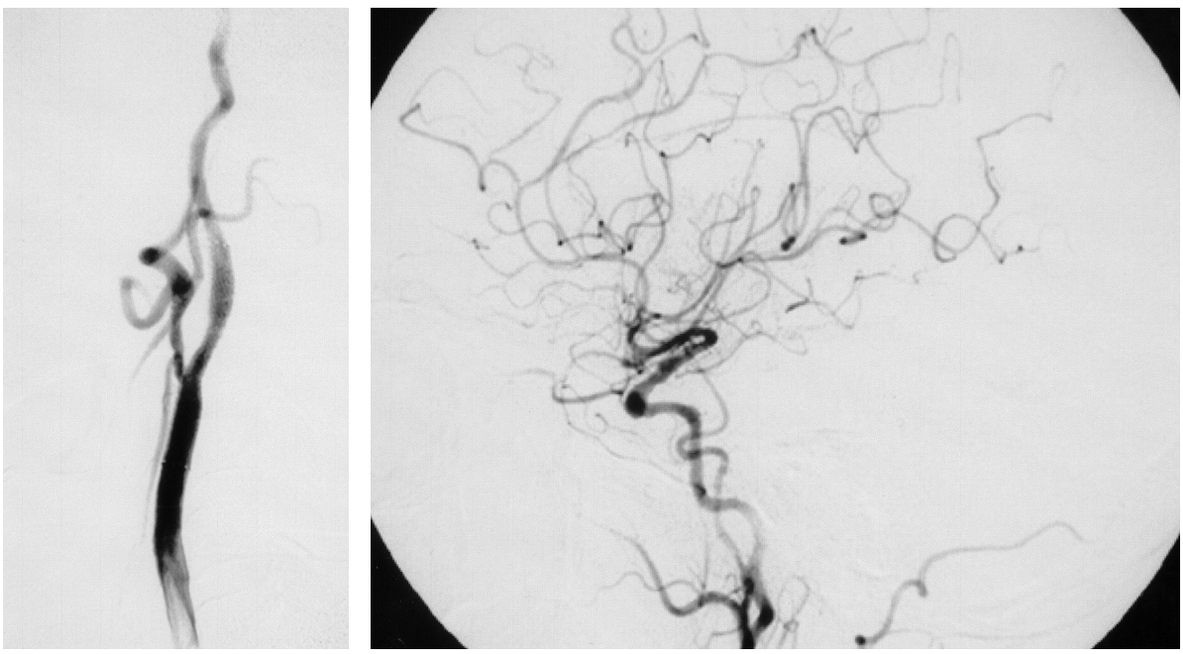


Figure 5 Lateral view of the left common carotid angiogram after treatment, showing dilation and antegrade blood flow in the left ICA.

ICA is unclear, the following mechanism may be considered. Fragmentation of atheroma generated by the guidewire manipulation may embolize severe stenotic lesions or cause arterial dissection of the ICA. A balloon placed at the origin of the external carotid artery may squeeze or rupture the atheroma into the ICA. After inflating the balloon proximal to a very tight stenosis, the minor degree of reversed flow may cause thrombosis. Whenever this protection device is utilized, the interventionist should be careful about the possibility of an acute occlusion of the site of stenosis. Further advances in the technology of devices such as reduced size and improved crossability of guidewires will provide more safety and adequate results in the treatment of severely stenotic carotid lesions.

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